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Amendments to the Claims:

This listing of claims replaces all prior versions and listings of claims in the application:

Listing of Claims:

1-26. (Canceled)

27. (currently amended) A method of producing insulin in a subject in vivo, the method comprising introducing into the subject an intermediate lobe pituitary cell that has been genetically engineered to express insulin comprises a nucleic acid sequence encoding insulin, the nucleic acid sequence being operatively linked to a heterologous promoter that directs expression of the nucleic acid sequence in the intermediate lobe pituitary cell.

28-29. (Canceled)

- 30. (previously presented) The method of claim 27, wherein said intermediate lobe pituitary cell is an autologous cell.
- 31. (currently amended) The method of claim <u>27</u> <u>28</u>, wherein said subject is a human and the intermediate lobe pituitary cell is an autologous cell.

Claims 32-59 (Canceled)

- 60. (previously presented) The method of claim 27, wherein said intermediate lobe pituitary cell is an allogenic cell.
- 61. (previously presented) The method of claim 27, wherein said intermediate lobe pituitary cell is a xenogenic cell.

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62-63. (Canceled)

64. (currently amended) The method of claim <u>27</u> 28, wherein said cell further comprises one or more nucleotide sequence encoding a protein that controls expression of insulin in a glucose stimulated manner.

- 65. (previously presented) The method of claim 64, wherein said protein that controls expression of insulin in a glucose stimulated manner is a glucokinase.
- 66. (previously presented) The method of claim 65, wherein said glucokinase is the β -cell isoform of glucokinase.
- 67. (previously presented) The method of claim 64, wherein said protein that controls expression of insulin in a glucose stimulated manner is a glucose transporter.
- 68. (currently amended) The method of claim 66 67, wherein said glucose transporter is GLUT-2.
- 69. (previously presented) The method of claim 64, wherein said protein that controls expression of insulin in a glucose stimulated manner is an ion channel that mediates glucosestimulated insulin release.
- 70. (currently amended) The method of claim 68 69, wherein said ion channel that mediates glucose-stimulated insulin release is a K+/ATP ion channel.
- 71. (previously presented) The method of claim 64, wherein said protein that controls expression of insulin in a glucose stimulated manner is glucagon-like peptide-1 (GLP-1).

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72. (previously presented) The method of claim 64, further comprising evaluating the subject for a parameter relating to glucose metabolism or insulin secretion.

- 73. (currently amended) The method of claim 74 72, wherein said parameter is selected from the group consisting of: the amount, distribution or structure of intracellular or extracellular insulin; glucose phosphorylating activity; glucose utilization; glucose uptake; and insulin secretion.
- 74. (currently amended) The method of claim <u>27</u> <u>28</u>, wherein said control region <u>promoter</u> is a pro-opiomelanocortin (POMC) promoter.

75-78. (Canceled)

- 79. (previously presented) The method of claim 27, wherein said intermediate lobe pituitary cell is a fetal or post natal cell.
 - 80. (previously presented) The method of claim 27, wherein said subject is a human.
- 81. (previously presented) The method of claim 27, wherein said intermediate lobe pituitary cell is a cultured cell.
- 82. (currently amended) The method of claim 80 81, wherein said cultured cell is a cultured human cell.
- 83. (previously presented) The method of claim 27, wherein said cell is from a non-human transgenic animal.

84-85. (Canceled)

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86. (previously presented) The method of claim 27, further comprising the step of administering an immunosuppressant to the subject.